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発表の概要と成果 (抄録を公開している URL がある場合、「概要・成果」を記載した上で、URL を末尾に記してください。また、抄録 PDF は別途ご提出ください。なお、抄録 PDF は Web 上には公開されません。)	
<p>Autophagy is a fundamental intracellular degradation system essential for maintaining cellular homeostasis, preventing diseases such as neurodegenerative and metabolic disorders, and contributing to anti-aging. Polyamines, including spermidine, spermine, and putrescine, are polycationic metabolites that are ubiquitously present in all living cells and regulate various biological processes, such as cell proliferation, differentiation, and gene expression. Extensive research has demonstrated that spermidine activates autophagy and extends lifespan in multiple model organisms, establishing the polyamine-autophagy axis as a promising therapeutic target for age-related diseases. However, despite numerous studies on spermidine, the functional characteristics of other polyamine compounds in the regulation of autophagy remain largely unexplored. Furthermore, a critical challenge in autophagy research in food science is the limited use of analyses employing true autophagic degradation activity (autophagy flux). Consequently, although many food components have been reported to affect autophagy, the precise nature of their impact remains largely unclear. In this study, we established an intestinal epithelial cell line stably expressing an autophagy-dependent fluorescent probe, enabling the quantitative measurement of autophagy flux. Using this system, we screened a diverse library of polyamine compounds with poorly understood cellular functions. This screening identified a novel polyamine with potent autophagy flux-enhancing activity that surpassed that of previously reported polyamines. Mechanistic analysis revealed that this polyamine activates autophagy via an mTORC1-independent pathway, demonstrating a novel regulatory mechanism that has not been previously reported. This finding elucidates a novel polyamine-mediated autophagy control mechanism and provides promising insights into health-promoting materials.</p>	

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